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Aetiology and Impact of Intra-abdominal Sepsis on Surgical Management.

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An overview of intra-abdominal sepsis is necessary at this time with new experimental studies and audits on management outcomes. The understanding of the pathophysiology of the peritoneum in the manifestation of surgical sepsis and the knowledge of the source of pathogenic organisms which reach the peritoneal cavity are crucial to the prevention of intra-abdominal infection. Recent advances in interventional techniques for peritonitis have significantly reduced the morbidity and mortality of physiologically severe complicated abdominal infection. Sepsis is an evolving process and the sequelae reflect the increasing severity of the systemic response to infection. Prevention entails early recognition, prompt resuscitation and early surgical intervention to abort the process. Sepsis represents a major clinical challenge and their management demands a multidisciplinary approach.

Introduction

Intra-abdominal sepsis is one of the most challenging situations in surgery and usually presents as peritonitis[1-5]. Gastrointestinal perforation, with leakage of alimentary contents into the peritoneal cavity, is a common surgical emergency and may have life-threatening sequelae. Duodenal and gastric ulcers remain the two most common perforations of the gastrointestinal tract due to the increased use of non-steroidal anti-inflammatory drugs (NSAIDS). It occurs most often in elderly patients with co-existent medical problems, who are at increased risk of post-operative complications[6].

The prevalence of *Helicobacter pylori* in the low social economic classes and associated poverty have increased the incidence of duodenal and gastric perforations in the developing world.[7,8] Small bowel perforations are less common and the rapid onset of severe pain, similar to a perforated peptic ulcer, may be absent. Many patients have a history of gastrointestinal disease (e.g. Crohn's disease, typhoid) or a systemic disease in which small bowel perforation is a recognized complication (e.g. connective tissue disorders, radiation enteritis, lymphoma), and these conditions occur at the site of disease within the bowel. Penetrating or blunt abdominal trauma accounts for about 40% of small bowel perforations[9] Diverticular disease, carcinoma, inflammatory bowel disease and appendicitis are the principal causes of colonic perforation. Diverticular disease and carcinoma account for about 80% of all colonic perforations which explains why 70% of perforations occur in the sigmoid colon. The caecum- the weakest part of the colon- accounts for a further 15% of colonic perforations, usually secondary to a distal obstructing annular carcinoma.[9,11]

The mortality of perforated viscus increases with delay in diagnosis and management[1-3,9-12]. The recently reported 12-fold variation in the 30-day mortality rate following emergency abdominal surgery in Britain ranged from 3.6% in the best performing hospital to 41.7% in the worst[4]. This would be alarming in the developing world but proves the persistent challenge of intra-abdominal sepsis. The mortality is lower when operations are conducted by consultant anaesthetists and surgeons rather than trainees and, where patients have ready access to treatment in intensive care following surgery. The 'surgeon factor' i.e. decision making on surgical management of the acute abdomen is the critical determinant of outcome[4]. 'Patient factor' is also important as most patients are over 65 with co-morbidity and often seriously ill with internal haemorrhage or a bowel perforation[4,13,14].

Perhaps the variation in surgical management outcome may also be partly explained by the demography and health of the local population. However, it is worth remembering that although most cases of infective

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(bacterial) peritonitis are secondary to gastrointestinal disease, it occasionally occurs without intra-abdominal sepsis (*primary peritonitis)* in ascites due to liver disease, haematogenous spread from a septic focus e.g. osteomyelitis in children, haemodialysis patients and the immunocompromised. Infections can also cause primary peritonitis, such as amoebiasis and candidiasis[15].

The acute abdomen

Surgical peritonitis and intestinal obstruction are the two important causes of the acute abdomen[16]. Surgical peritonitis may emanate from perforation, ischaemia (mesenteric or strangulation) pancreatitis and anastomotic leakage. Acute appendicitis is the most common cause of the acute abdomen requiring surgery with a life time risk of ~7%, and it is one of a relatively dwindling number of conditions in which a decision to operate may be based solely on clinical findings[10]. The aim of both the history and examination is to determine a diagnosis and clinical decision. It remains the ability to identify the presence or absence of peritoneal inflammation which probably has the greatest influence on the final surgical decision[7]. There are undoubtedly specific features associated with all acute abdominal conditions which are well established. Conditions that start suddenly and produce signs of peritonitis are perforation of viscus (e.g. peptic ulcer, typhoid) infarction (embolus or volvulus) and intraperitoneal haemorrhage (e.g. ruptured ectopic pregnancy, aortic aneurysm). Abdominal tenderness, due to intraperitoneal blood, has a different character and is less pronounced than that of peritoneal inflammation due to sepsis.

Conditions that produce peritonitis of gradual onset usually arise from a progressively inflammed viscus e.g. acute appendicitis/cholecystitis/diverticulitis[7-11]. Intra-abdominal abscesses may also occur within an intra-abdominal organ. These include pyogenic abscess in the liver from portal pyaemia when in a septicaemia organisms and neutrophil polymorphs embolize to the liver e.g. following appendicitis or a perforation (now fortunately rare because of the use of antibiotics); pancreas from acute pancreatitis, and in the fallopian tube (pyosalpinx) following adhesions in the fimbrae from an ascending infection[17]. Infections above obstructing calculi may include an empyema of the gallbladder or in the renal pelvis. Regular re-assessment of patients and making use of the investigative options available will meet the standard of care expected by patients with acute abdominal pain[7].

Sepsis is an *evolving* process. It is the systemic inflammatory response to infection frequently associated with hypoperfusion followed by tissue injury and organ failure. Therefore, its sequelae reflect increasing severity of the systemic response to infection and *not* severity of infection[1-3]. Thus mortality increases with the degree of the systemic inflammatory response syndrome (SIRS). The mortality following a *bacteraemia* is 5%, *sepsis* (infection + SIRS) is 15%, *septic shock* (sepsis+ hypotension (systolic BP< 90mmHg) ~50%, severe *SIRS* 80%, *multiple organ failure* (MOF) 90% [1-3]. SIRS is a massive systemic response comprising an evolution of a cytokine cascade (TNF, IL-1. IL-6, IL-8), and a sustained activation of the reticulo-endothelial system. It finally leads to the elaboration of secondary inflammatory mediators causing cell damage. These mediators include arachidonic metabolites (prostaglandins and leukotrienes), nitric oxide (vasodilator), oxygen free radicals, platelet activating factor causing increase platelet deposition, vasodilatation, increase capillary permeability and activation of coagulation pathways which results in end -organ dysfunction by formation of microthrombi. In multiple organ dysfunction syndrome, the mortality rates in intensive care units increases with the number of organs failed ; a 40% mortality for single organ failure, 60% for 2 organ failure and 98% for 3 organ failure.[5] Early goal-directed resuscitation during the first 6 hrs after recognition of shock has moved towards the use of whole blood as it appears to eliminate the problems of expansion of extravascular volume seen with crystalloid and also appears to provide a lower incidence of organ failure[18].

Bacteria synergism

Most cases of peritonitis are caused by organisms derived from the gastrointestinal tract i.e. endogenous[9-11]. The contents of the stomach and duodenum are more sterile than the contents of the distal gut. Thus, the sequelae of an upper gastrointestinal tract perforation are less severe than that of lower gastrointestinal

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tract at least in the early stage. The peritoneal fluid is initially sterile due to host defense mechanisms but secondary bacterial invasion occurs within 6 hours and bacterial peritonitis follows chemical peritonitis. Infection is enhanced by the synergy between aerobes e.g. *Escherischia col*i which reduce oxygen content and facilitates growth of obligate anaerobes e.g. *Bacteroides fragilis*, and by the presence of adjuvant substances e.g. faeces, bile or urine[23]. Untreated, colonic perforation with faecal peritonitis is rapidly fatal because of the absorption of this pathogenic bacteria load and their toxins from the peritoneal cavity causing septicaemia[24]. This produces a rapid and profound systemic inflammatory response syndrome (SIRS) with consequent multiple organ failure to which the elderly easily succumbs[3,5].

''*Source control*'' is defined as any procedure, or series of procedures, that eliminates infectious foci, controls factors that promote on-going infection, and corrects or controls anatomic derangements to restore normal physiologic function[19]. Source control failure is more likely in patients with delayed (> 24 hours) procedural intervention, higher severity of illness (Acute physiology and chronic health evaluation score or APACHE >15), advanced age (> 70 years), co-morbidity, poor nutritional status, and a higher degree of peritoneal involvement, and is heralded by persistent or recurrent intra-abdominal infection, anastomotic failure, or fistula formation[20].The peritoneum comprises a serous membrane made of mesothelial cells lining the abdominal viscera (visceral layer) and separating it from the surrounding abdominal wall (parietal layer). The parietal and visceral parts are in continuity around the root of the viscus and are separated from each other by a cavity which normally contains only a thin fluid of serous fluid. This permits movement between the viscus and its surroundings[25]. The peritoneum has a large surface area ($2m^2$) almost equivalent to the total body surface area. Its semi-permeable membrane allows rapid two-way passive fluid transport of water and most solutes, and, the specialized lymphatics in the diaphragm actively absorb bacteria, fluid, particles and deformable particles as large as leucocytes. In the normal peritoneum there is rapid movement of fluid, bacteria and leucocytes along well- defined pathways around the peritoneum, through the diaphragmatic lymphatics to the mediastinal lymphatics and thence to the thoracic duct. This *dispersion* of infection is facilitated by the fibrinolytic activity of the peritoneum derived from mesothelial cells and submesothelial blood vessels. However, this activity is lost even after minor peritoneal injury resulting in rapid adhesion between affected surfaces. Therefore, peritoneal resistance depends on *localization* rather than dispersion[26].

Manifestations of intra- abdominal sepsis

A complicated abdominal infection extends beyond the hollow viscus of origin into the peritoneal space and is associated either with abscess formation or peritonitis.

Localized peritonitis thus occurs because peritoneal resistance to infection relies upon localization rather than dispersal of a contaminant. The inhibition of peritoneal fibrinolysis permits stabilization of fibrinous exudates and limits the spread of infection. The omentum 'abdominal policeman' and the intraperitoneal viscera also have a remarkable ability to confine infection as seen for example in acute appendicitis, perforated duodenal ulcer/ diverticular disease[28, 29]. Localised peritonitis implies either contained or early perforation of a viscus or inflammation of an organ in contact with anterior parietal peritoneum. For instance, a palpable mass in the right iliac fossa represents either an inflamed mass of adherent omentum, appendix and adjacent viscera, or an abscess. Conservative treatment with later drainage of any abscess had been the standard and diffuse peritonitis was usually fatal. Surgery for appendicitis evolved when the mortality associated with perforated appendicitis was high. The prognosis after surgery is excellent[10, 28]. Although only a few patients progressed to the potentially lethal complications, early surgery for all patients with suspected appendicitis became the definitive method of preventing severe peritoneal sepsis[10].

Generalized peritonitis will occur when there is failure of localization. Failure of localization may arise for the following reasons: a) a rapid contamination that does not permit localization as in a perforated colon/ anastomotic leak, b) persistent or repeated contamination that overwhelms an attempt to overcome it, c) a

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localized abscess that continues to expand and ruptures into the peritoneal cavity (e.g. appendix, diverticular abscess)[24,28]. The peritoneal cavity becomes acutely inflamed with production of an inflammatory exudate which spreads through the peritoneum leading to intestinal dilatation and paralytic ileus.

1) Acute colonic perforations.

The risk of generalized peritonitis secondary to appendicular perforation is greater in children, in whom the omentum is poorly developed, and the elderly, in whom vascular occlusion and gangrenous appendicitis are more likely[28]. Perforation, is the most common complication of diverticular disease. Initially, a pericolic abscess forms, which subsequently ruptures into the peritoneal cavity with the development of generalized and sometimes faecal peritonitis[9]. It can be difficult to differentiate diverticular and stercoral perforations because the two often coexist in the sigmoid area. Stercoral perforation is caused by an ischaemic pressure necrosis of thecolonicwallfromimpactesolidfaeces[9,11].

In patients with a colonic carcinoma, large bowel perforation may occur at the site of the tumour due to invasion of surrounding structures, with initial abscess formation followed by free perforation. These tumours are locally advanced, may be unresectable and over 50% of patients have hepatic metastasis at the time of presentation. Perforation may complicate a toxix megacolon in infective or ulcerative colitis but is uncommon in patients with a toxic megacolon secondary to Crohn's disease[11].

It may be difficult to reach the correct pre-operative diagnosis in many patients with colonic perforations. As patients often present with generalized peritonitis, the diagnosis is being established only at laparotomy[7,9]. There may be clues in the history e.g. known diverticular disease, ulcerative colitis, Crohn's disease, collagen disorder; a recently altered bowel habit or rectal bleeding suggesting carcinoma, or there may be a short history of severe diarrhoea in patients with infective colitis.[11] Patients with faecal peritonitis are more severely ill with signs of septic shock (dehydration, oliguria, hypotension, peripheral circulatory failure, hypothermia, cerebral disorientation) than those with a purulent peritonitis from a small perforation[2,3].

Surgical implications

A laparotomy should be performed once the patient has been resuscitated, and has been given opiate analgesics and intravenous antibiotics. A vertical midline incision gives access to the entire abdomen and the perforation site should be identified and the aetiology determined. As the most common operative finding in patients with a colonic perforation is a perforated inflammatory mass in the sigmoid colon, differentiation between diverticular disease and carcinoma may be difficult[9]. Current opinion favours resection of the inflammatory phlegmon and its perforation, with or without primary anastomosis[11]. The former policy of peritoneal drainage, construction of a proximal defunctioning stoma and subsequent colonic resection at a second operation carries a postoperative mortality of 30%[9, 13]. The stoma does not protect against continued faecal contamination from the perforated segment. Thus, the importance of 'source control' of sepsis[19]. Many surgeons favour a Hartmann's procedure with excision of the diseased bowel, construction of an end colostomy and closure of the rectum, or alternatively exteriorization as a mucous fistula[24]. Patients with a toxic megacolon require a subtotal colectomy with preservation of the rectal stump and formation of a terminal ileostomy. This allows the option of construction of an ileo-anal pouch once the sepsis has resolved[9, 11].

Light clothing, hot climate, high residue diet (vegetables) and poor availability of appliances all make the management of an ileostomy more difficult in the tropics and so ileorectal anastomosis is preferred unless the rectum is extensively diseased with stricture formation. Careful resuscitation inorder to avoid congestive heart failure from the toxic myocarditis of typhoid and prompt surgical intervention has reduced the mortality rate from typhoid perforation from 50% to 20%. The perforated ulcer should be excised by wedge excision, a single area of diseased bowel may be resected or, in the very ill patient, exteriorization of the small bowel may be the best procedure[8].

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2) Anastomotic leakage

Anastomotic leakage may be early (3-5 days post operation) as a result of technical failure, or late (weeks) as a result of biological failure e.g. due to ischaemia or inherent disease. Interestingly, postoperative mortality from post operative sepsis due to anastomotic leak is higher than any natural condition[29]. The mortality rate of individuals who developed an anastomotic disruption was 39.3%, and anastomotic leak was found to be an independent predictor of mortality[30]. This may be due to the fact that sepsis is the leading cause of death following an anastomotic leak and corroborated by the fact that delayed diagnosis worsens the prognosis. The acute onset of abdominal pain and generalized peritonitis is a serious manifestation of an anastomotic leak and, these patients may quickly progress to septic shock, requiring intensive care monitoring and resuscitation with fluids and inotropic agents[1, 2.] In general, either a leak presents as localized, being walled-off by omentum and small intestine, and presenting itself as an intra-abdominal abscess, or less frequently, a leak presents as a free perforation with faeculent peritonitis. Patients with diffuse peritonitis from an anastomotic leak or perforated viscus cannot be fully resuscitated until ongoing soiling has been controlled[19].

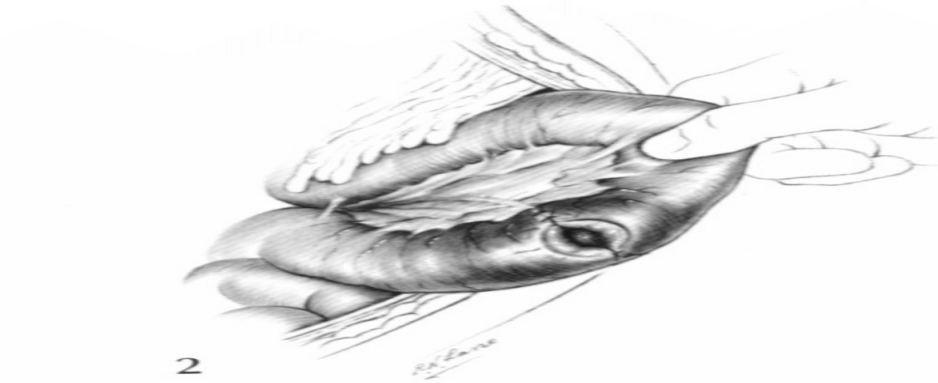

Figure1. Anastomotic leakage[36]

In such patients resuscitation should be continued intraoperatively with exteriorization of bowel ends as stomas[11, 24, 31]. Laparostomy as opposed to primary closure of abdominal fascia may be indicated if there is a risk of developing an abdominal compartment syndrome from severe sepsis and septic shock[32]. For haemodynamically stable patients without generalized peritonitis e.g. abscess, a delay of up to 24 hrs may be appropriate to allow further clinical assessment and image-guided minimally invasive interventional therapy[20,21]. A delayed manifestation of anastomotic leak as a colocutaneous fistula is rare[30]. Despite modern surgical techniques and significant improvements in intra- and peri-operative care of the surgical patient, the colorectal anastomosis still has an anastomotic leak rate reported to range from 3% to 22%[29]. Prevention of sepsis from adequate attention to technique, and correct surgical decision making from the findings at operation, as to the right operative procedure for each patient (i.e. surgeon- related factor) remains the single most important factor that can influence the morbidity and mortality in bowel surgery[11,13,24,29].

3*) Intestinal (enterocutaneous) fistula* is usually a confined intestinal leakage with no generalized peritonitis. Abdominal fistulas often arise from a septic process and always have the potential to create a septic process. It may be *spontaneous* from a perforated diseased viscus e.g. from intestinal Crohn's, tuberculosis, typhoid etc; or *iatrogenic* from an anastomotic leak or intraoperative bowel injury. It may be simple (a lateral end fistula) or complicated (complete disruption of bowel ends, distal obstruction, complex abscess cavity, diseased bowel, mucocutaneous continuity etc) [33].

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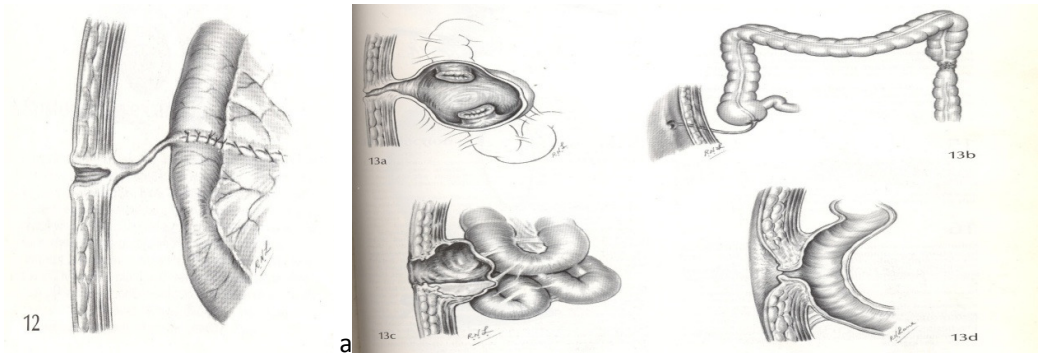

Figure 2. Simple and complex enterocutaneous fistulae[33]

It may be high output (>500mls/24hrs) from proximal small bowel with a more severe physiological sequelae (with respect to fluid, electrolytes and nutritional derangement), or low output (<500mls/24hrs) from distal small bowel and colon. A simple iatrogenic fistula should heal with conservative management but a complex fistula will require surgical intervention[34].

Complications of generalized peritonitis

Intra-abdominal (intraperitoneal) abscess can occur as a result of a localized perforated viscus, or a generalized peritonitis that fails to resolve completely and becomes localized, or, as a post operative complication from an anastomotic leak or inadequate elimination of abdominal sepsis[13].

i) ***Localized perforated viscus.*** The perforation rate of acute appendicitis is 25% in patients with a history of pain of less than 24 hrs or 35% in patients with a history over 48 hrs[10]. There is no evidence to indicate the proportion of patients likely to develop diffuse sepsis because early surgery for all patients with suspected appendicitis became the definitive method of preventing severe peritoneal sepsis[7]. Because the perforation is sealed rapidly by omentum, the signs remain localized. There may be a mass but unlike an appendix mass, the patient is systemically unwell with significant abdominal tenderness. The inflammatory phlegmon/mass may also cause an extrinsic distal small bowel obstruction. This condition is best treated by surgical intervention through a standard right iliac fossa incision. A residual necrotic appendix usually found can be resected and adjacent friable tissue and organs handled with care[27]. Saline or antibiotic lavage of the abscess cavity and wound is a simple and effective method of preventing residual infection[35, 36]. Peritoneal and wound drains are of no use[11,13,35]. The importance of surgical access is corroborated by the finding of a 40% wound sepsis rate, by using a small incision to remove a perforated appendix[37]. Restricted exposure and access lead to inadequate surgery with incomplete peritoneal toilet and lavage and limited colonic mobilization. Interestingly, studies suggest that whereas surgery may be associated with adhesions, subsequent tubal infertility is only adversely affected in patients with perforated appendicitis[38]. Percutaneous drainage may be used in complex settings including infected necrotizing pancreatitis even in the presence of organ failure as by removing the necrotic tissue/abscess and decreasing pancreatic tissue tension prevents the progression of SIRS[19,20]. Operation may also be necessary to exclude malignancy (lymphoma) or TB ileitis where a chronic appendiceal abscess/phlegmon present as a palpable mass in a patient with a history of fever, night sweats, tender adenopathy and weight loss [10, 11].

ii) ***Post operative intra-peritoneal abscess.*** Is commonly due to inadequate elimination of sepsis despite all efforts especially following generalized faecal peritonitis[13, 24]. The main sites of intra-abdominal abscess are usually over the site of the origin of infection or in the dependent areas of the body: subphrenic spaces, pelvis (pouch of Douglas), hepatorenal (Morrison's) pouch, paracoloc gutters and the lesser sac. When the

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body is lying supine, the posterior abdominal wall is higher in the iliac fossa than behind the liver so peritoneal fluid gravitates behind the liver along the paracolic gutters. Clinical suspicion of a post operative intra-abdominal abscess will include a swinging pyrexia, increasing pain, pulse and mass[7]. Unlike generalized peritonitis which demands emergency laparotomy, intra-abdominal abscess must be treated urgently, not emergently. Drainage should be performed within 12 h of diagnosis, but, patients critically ill with a severe systemic septic response require immediate drainage following initial haemodynamic and respiratory resuscitation[14, 39]. CT or Ultrasound – guided percutaneous drainage of abdominal abscesses has emerged as the procedure of choice in many circumstances as morbidity and mortality is lower than following operative drainage[40]. A morbidity rate of 16% v 4% and mortality rate of 21% v 10% is noticed in one series[41]. Operative drainage is necessary for those abdominal abscesses which are multiple, are isolated but cannot safely be approached percutaneously, and/ or are associated with systemic sepsis unresponsive to percutaneous drainage[30, 41].

A localized pelvic abscess may be drained through the rectum[30]. Postoperative peritoneal sepsis may be diffuse and result in paralytic or adhesive small bowel obstruction protracting convalescence and thus require operation. The mortality from post operative intra-abdominal abscess is greater than 50% and the mortality increases with each operation to treat recurrent or persistent sepsis[30]. Therefore, the best opportunity to eradicate infection is the first operation.

Prevention of intra-abdominal sepsis

It is not possible to practice fully the ideal management of early diagnosis and surgery for the acute abdomen, thus reducing morbidity and mortality to zero, because patients and the disease are variable. However, because infection, inadequate tissue perfusion and a persistent inflammatory state are the most important risk factors for development of multiple organ failure it seems logical that initial therapeutic efforts should be directed at their early treatment or prevention (early goal-directed therapy)[5]. Early definitive primary or reoperative surgery leading to the removal of necrotic tissue, the drainage of abscesses, and the control of peritoneal soilage (source control) may be effective in the intraabdominal septic patient[19]. Early initiation of broad spectrum antibiotics has been shown to be critical during the SIRS phase for prevention of sepsis and septic shock[22]. Critically ill patients who are either physiologically unstable or at high risk of failed source control especially following septic shock where resuscitation with crystalloid will likely lead to an abdominal compartment syndrome may benefit from a laparostomy[31,32]. Ongoing intestinal ischaemia or multiple areas of intestinal ischaemia occurring in patients with connective tissue disorders (e.g. polyarteritis nodosa) is best managed by a planned re-exploration at 24hrs later if there is any doubt or difficulty in assessing intestinal viability[9]. Diffuse peritonitis from perforated appendicitis, which has been diagnosed preoperatively, should be dealt with by formal laparotomy, rather than by making a gridiron incision, to allow thorough peritoneal toilet and lavage[27]. Untreated pockets of infected peritoneal fluid and failure to remove faecoliths cause postoperative sepsis.

However, despite improvements in resuscitation techniques, antibiotic therapy and anaesthesia, the mortality associated with a perforated peptic ulcer has not changed over the last two decades. It remains around 25% almost certainly due to the fact that the age-mix of the disease has changed during this time with more elderly (female) patients presenting with perforated peptic ulcers many of whom have serious concomitant medical illnesses (poor American association of Anaesthesia score or ASA) [6]. Similarly, the mortality of appendicitis is associated with the age of the patient and delayed diagnosis. The overall mortality for appendicitis is less than 1%, but it rises to over 5% when perforation is present.[10] Mortality is related to age as most deaths occur in the elderly[27]. The most important prognostic factors in emergency colorectal surgery are age and faecal peritonitis. Together the mortality is greater than 60%[24]. Thus peritoneal sepsis is seldom the sole cause of death, but it compounds coincidental cardiovascular, respiratory or renal pathology. The prevention of the progression of sepsis is by early goal-directed therapy and source control. When severe SIRS is in progress, prognosis is poor and surgical intervention may be late

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as the cascade is fully in progress. Supportive treatment may be all that is required as there is as yet no known drug to abort this cascade[3, 5].

Conclusions

The understanding of the pathophysiology of the peritoneum in the manifestation of surgical sepsis, and knowledge of the source of pathogenic organisms which reach the peritoneal cavity are crucial to the prevention of intra-abdominal surgical infection.

The ability to identify the presence or absence of peritoneal inflammation probably has the greatest influence on the final surgical decision.

Large bowel perforation carries a high risk of postoperative morbidity and a significant risk of mortality, even after expeditious and appropriate surgical treatment. The mortality of perforated viscus increases with delay in diagnosis and management. It is greatest in the elderly and those ill from intercurrent disease (poor ASA score).

Early recognition, prompt resuscitation and early surgical intervention will abort the evolving process of sepsis. More research on the pathophysiology of sepsis are required to improve mortality and morbidity in these critically ill patients.

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